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Research paper

Acoustic stimulation of human medial olivocochlear efferents reduces stimulus-frequency and click-evoked otoacoustic emission delays: Implications for cochlear filter bandwidths

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ABSTRACT

Filter theory indicates that changes in cochlear filter bandwidths are accompanied by changes in cochlear response latencies. Previous reports indicate that otoacoustic emission (OAE) delays are reduced by exciting medial olivocochlear (MOC) efferents with contralateral broad-band noise (CBBN). These delay reductions are consistent with MOC-induced widening of cochlear filters. We quantified the MOC-induced changes in human cochlear filter-related delays using stimulus-frequency and click-evoked OAEs (SFOAE and CEOAEs), recorded with and without MOC activity elicited by 60 dB SPL CBBN. MOC-induced delay changes were measured from the slopes of SFOAE phase functions and from cross-correlation of 500 Hz-wide CEOAE frequency-band waveform magnitudes. The delay changes measured from CEOAEs and SFOAEs were statistically indistinguishable. Both showed greater delay reductions at lower frequencies (a 5% decrease in the 0.5–2 kHz frequency region). These data indicate that cochlear filters are widened 5% by the MOC activity from moderate-level CBBN. Psychophysically, the large changes in cochlear response latencies, implied by the 0.5 ms change in OAE delay at low frequencies, would have a profound effect on binaural localization if they were not balanced in the central nervous system, or by the MOC system producing similar changes in both ears.

1. Introduction

One of the fundamental properties of the auditory system is the ability to analyze sounds into different frequency components, a process usually conceptualized as being done by a bank of cochlear filters. The frequency resolving ability of the cochlea is determined by its filters' bandwidths. However, cochlear filter bandwidths are not fixed. Indeed, their bandwidths increase at high sound levels as shown by tuning curves (TCs) from basilar-membrane (BM) motion or auditory-nerve (AN) responses (e.g. Temchin et al., 2008). This happens because cochlear amplifier (CA) gain, which is greatest near the peak of the filter, decreases as sound level increases. CA gain is also decreased by stimulation of medial olivocochlear (MOC) efferent neurons (Guinan, 1996), so MOC activity would be expected to widen cochlear filters. In cats, Guinan and Gifford (1988) found that electrical MOC stimulation widened TCs for AN fibers with CFs greater than 2 kHz. Both increases and decreases in bandwidth were found for fibers with CFs less than 2 kHz. Contralateral acoustic stimulation (CAS) can also be used to elicit MOC activity that reduces CA gain and inhibits AN responses (Warren and Liberman, 1989a, b). Thus, CAS-elicited MOC activity would also be expected to widen cochlear filters.

Filter theory indicates that widening a filter produces a shorter impulse response (Oppenheim and Wilsky, 1997). Thus, widening cochlear filters would be expected to shorten cochlear response latencies. In humans, otoacoustic emissions (OAEs) provide a non-invasive measure of cochlear mechanical responses. Stimulus-

Abbreviations: CBBN, contralateral broad-band noise; OAE, otoacoustic emission; SFOAE, stimulus-frequency otoacoustic emission; CEOAE, click-evoked otoacoustic emission; DPOAE, distortion product otoacoustic emission; SOAE, spontaneous otoacoustic emission; CA, cochlear amplifier; CAS, contralateral acoustic stimulation; BM, basilar-membrane; MOC, medial olivocochlear; TC, tuning curve; AN, auditory-nerve; CF, characteristic frequency; SNR, signal-to-noise ratio; MEM, middle-ear muscle; STFT, short-time Fourier transform; FFT, fast Fourier transform; RMS, root-mean-square; WT, wavelet transform; ANOVA, analysis of variance; pTC, psychophysical tuning curve; ITD, interaural time difference.

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frequency OAEs (SFOAEs) and click-evoked OAEs (CEOAEs) are both thought to be produced by a single process, coherent reflection (Shera and Zweig, 1993), in which OAE delays are inversely related to cochlear filter bandwidths. Under this premise, an MOC-induced widening of cochlear filters should be seen as reduced CEOAE and SFOAE delays. Human distortion product OAE (DPOAE) delays are less suitable for inferring changes in cochlear filter bandwidths because they are the mixture of two components with different delays (Shera and Guinan, 1999; Kalluri and Shera, 2001; Withnell et al., 2008), and DPOAE phase-gradient delays are highly parameter dependent (Shera et al., 2000), which obscures measurement of MOC-induced changes in cochlear filter-related delays.

There are several reasons for being interested in the delays that MOC activity may produce in cochlear responses, aside from what these delays tell us about MOC-induced changes in cochlear filter bandwidths. First, interaural delay is one of the primary cues for sound localization and MOC-induced changes in cochlear response latencies may affect interaural delays. Second, human psychophysical frequency selectivity is not simply explained by cochlear filtering (Cedolin and Delgutte, 2005). The timing of AN responses also contains information about the frequency content of a sound. One theory hypothesizes that a cochlear traveling wave produces phase differences across AN fibers of different CFs and that these phase differences provide an important cue for resolving the frequency components in a complex sound (Shamma, 1985; Carney, 1992). An MOC-induced change in cochlear response latencies will affect these phase differences and may therefore affect cochlear frequency selectivity by changing the timing of AN responses, as well as by changing filter bandwidths.

There have been many previous studies of MOC effects on OAEs, but most reported only the change in OAE amplitudes and few reported MOC-induced changes in response phase or delay. Ryan et al. (1991), Berlin et al. (1993) and Giraud et al. (1996, 1997) reported that CAS-elicited MOC activity decreased OAE delays, and perhaps more so at lower frequencies. In our study, we refined the OAE-based methodology to provide a more detailed and accurate description of MOC-induced OAE delay changes.

To quantitatively determine the changes in OAE delays produced by MOC activity, we measured SFOAE phase-gradient delays and CEOAE latencies, with and without MOC activity elicited by CAS. Since SFOAEs and CEOAEs are thought to be due to the same underlying mechanism (Shera and Guinan, 1999; Kalluri and Shera, 2007), they should show the same MOC-induced delay changes.

2. Materials and methods

2.1. Methods overview

MOC-induced changes in OAE delays were calculated from the comparison of OAEs obtained with and without MOC activity elicited by contralateral noise. For SFOAEs, changes in cochlear delays were measured from the changes in phase-gradient delays obtained from the slopes of linear fits to SFOAE phase versus frequency data. For CEOAEs, changes in cochlear delays were measured by separating the click responses into 500 Hz-wide frequency regions, and in each frequency region, cross-correlating the CEOAE waveform magnitudes with and without MOC activity.

2.2. Stimuli and acoustics

To evoke SFOAEs, a 40 dB SPL, unilateral probe-tone was presented continuously in the test (ipsilateral) ear. Ear-canal sound pressure was averaged over 5.3 s repeated time periods called "trials". In each trial, there was an initial 500 ms with the probe-tone alone to provide a "baseline," (see below) followed by the concurrent presentation of a 2.5 s MOC elicitor, which was a 60 dB SPL broad-band noise in the contralateral ear. Starting 600 ms before the end of the trial, a 500 ms, 55 dB SPL suppressor tone, 50 Hz higher in frequency than the SFOAE probe-tone, was presented to the ipsilateral ear (see below). Both the elicitor and the suppressor were alternated in polarity across trials so their acoustic waveforms (but not their effects) would cancel in averages. We refer to the sequential presentation of trials with opposite polarity stimuli as, "alternations." To provide data from which phase-gradient delays could be calculated, SFOAE measurements were done at 3–7 probe-tone frequencies, spaced 20 Hz apart, in a random order. We averaged ear-canal response waveforms in sets of 8 trials (4 alternations) per probe-tone frequency. We refer to a set of trials that was averaged as a "recording block." The number of recording blocks for each phase-gradient delay measurement varied, depending upon subject availability and the number of probe-tones in a frequency group, but on average there were 7 recording blocks for each phase-gradient delay measurement. For each subject, the SFOAE test frequencies were selected by initially obtaining CEOAE data and then choosing frequencies at CEOAE spectral peaks with large signal-to-noise ratios (SNRs). Frequencies within 50 Hz of a spontaneous OAE (SOAE) greater than −10 dB SPL were not used in order to avoid unintended acoustic interactions within the cochlea.

To evoke CEOAEs, 100 μs clicks were presented at 50 dB pSPL and a rate of 40 Hz. 40 Hz was used (instead of the standard 50 Hz) to reduce unintended MOC activity elicited by the clicks (Veuillet et al., 1991; Guinan et al., 2003). The ear-canal sound pressure was digitally sampled at 100 kHz (to enhance temporal accuracy) and responses were averaged in 500 ms repeated time periods or "trials." The responses in the first trial period were omitted (to allow time for build-up or decay of the MOC effect) and then 128 trials were averaged (called a "block"). After averaging, each block was divided into the 25 ms segments containing the CEOAEs. These segments were averaged, so that a block average contained 2560 CEOAEs. Typically, three blocks without the CAS elicitor and three interleaved blocks with the CAS elicitor were averaged for each ear. This resulted (typically) in 7680 CEOAEs averaged per CAS condition. We averaged this large number of responses to maximize CEOAE SNRs. To obtain a noise floor, the same number of trials were averaged without any sound.

For all experiments, various tests were done to ensure that the recorded changes were due to MOC effects. Data collection trials containing large response artifacts due to subject movement were excluded before averaging. Post-averaging processing included further rejection of averages containing abnormally large transient responses missed during online artifact rejection, and averages in which ear-canal pressure showed large differences between the beginning and end of a trial. For each ear, a suppressed-SFOAE middle-ear muscle (MEM) test was done (Lilaonitkul and Guinan, 2009a). These tests did not show any MEM contractions produced by the CAS MOC elicitor for any subject. Although click-trains can elicit MEM contractions, a 40 Hz rate and 50 dB pSPL level are most likely too slow and low-level to elicit substantial MEM activation (Guinan et al., 2003).

Ear-canal sound pressure was recorded using Etymotic Research ER10c acoustic assemblies in each ear. The outputs from the two ER10c acoustic sources were calibrated in each ear at the beginning and frequently throughout each recording session. Each acoustic stimulus was produced by a separate earphone to avoid nonlinear stimulus interactions in an earphone. The broad-band noise (0.1–10 kHz) used to elicit MOC activity was spectrally flattened in each subject using in-ear acoustic calibrations.